Determinants of pericardial drainage for cardiac tamponade following cardiac surgery†

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We have read the interesting article by Pompilio et al. [1] about the determinants of the pericardial drainage for cardiac tamponade. It is a well-designed analytic paper that can be useful for everyday practice, however; some issues have to be addressed. First, did tamponade develop during hospitalization or after the discharge of the patient? Did any of the patients have any surgical bleeding? Secondly, the authors have reported the revision rate as 4.2%. We have previously published a paper about the postoperative re-exploration for bleeding [2]. In a patient population more than 19,000, we had 1.4% revision for bleeding which is compatible with the literature. How do the authors explain the relatively high rate of need for revision for bleeding postoperatively? Thirdly, in Table 2, it is stated that 1.8% of the patients had anticoagulant usage. How many of these patients were in the pericardial drainage group? Were there any abnormalities in the liver function tests and was there any difference between the groups? Could the authors give more details about the utilization of antifibrinolytic and clotting factors in their centre? Lastly, in the Statistical methods section, it is stated that the univariate tests were run three times. Were there any differences between these three runs of tests? We would like to thank the authors for their study and would like to learn about their comments on the subject.

REFERENCES


LETTER TO THE EDITOR

Severe intraprosthetic regurgitation by immobile leaflet after transcatheter aortic valve implantation

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We read with interest the case report by Al-Attar et al. [1] regarding the fatal complication of severe intravalvular leak due to an immobile cusp following trans-apical TAVI with 23 mm Edwards SAPIEN valve. We congratulate the team for sharing their complication which is under reported and at times overlooked. The patient developed rapid severe left ventricular dys-function despite all attempts to correct the situation failed (including positioning of a second valve-in-valve and the use of femorofemoral Extracorporeal membrane oxygenation). We also noted the editorial comment regarding the possibility of damage to the valve leaflets while hooking the leaflet on the stent during the crimping phase as a possible origin of the irreversible immobility.

At our institute, over the past three years, we have implanted over 230 Edwards SAPIEN and XT valves [2]. We have also noted the onset of this complication in two patients. The immobility of the stent valve leaflet was transient and due to inadequate balloon inflation of the valve in the patients. Once the valve is balloon expanded again, the stent fully deployed allowing the valve leaflets to function properly. We do not believe that crimping is the cause but as described by the authors it must be an irregular expansion on the stent, which leads to aortic regurgitation. We think noncircular expansion is the major mechanism, as implanted stented surgical valves are circular and always function well unless there is distortion of the stent during implantation. Also, aortic regurgitation is observed after stentless valve implantation when there is distortion of the stentless valve anatomy, i.e. loss of circular shape. Hence they need expertise and experience to achieve best results [3]. Once implanted the valve opens due to ventricular contraction and closes due to the eddy currents generated [4]. If with adequate pressure head the leaflet function is not satisfactory, then we agree that the only option is implanting another stent with or without circulatory support depending on the degree of regurgitation and haemodynamic stability. In our centre, we performed a case where valve-in-valve implantation was necessary to achieve successful outcome after such a complication. In our experience, we believe it is unlikely that crimping is a cause for this complication.

The published literature suggests complications related to the valve mechanism for the Edwards SAPIEN valve, and valve-in-valve implantation or implantation of a second valve being 2.6% [5]. Bearing these observations in mind, we too support the editorial reflections in strongly advocating cardiovascular surgical team present jointly for all TAVI cases, with the facility to go on cardiopulmonary bypass expeditiously. In our experience the procedure is performed under general anaesthesia with continuous transoesophageal echocardiography. This allows excellent haemodynamic control and visualization of the valve with early identification of the mechanism of valve dysfunction and treatment. In order to expand the application of this technology to moderate risk populations in the near future, we need to ensure that TAVI is performed in highly controlled environment. There needs to be anaesthetic, echocardiographic and surgical team support to lower complications, reduce mortality and achieve excellent outcomes.

**REFERENCES**


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**LETTER TO THE EDITOR RESPONSE**

**Reply to Attia and Bapat**

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**Keywords**: Aortic value • TAVI • Complications

We thank Drs Attia and Bapat for their comments and for sharing their experience. The analogy the authors make with stentless valve implantation is quite logical and supports the hypothesis of non-circular expansion of the deployed valve. [1] This complication is uncommon but illustrates how poorly tolerated acute aortic regurgitation (AR) is after transcatheter aortic valve implantation (TAVI). Indeed unless rapid deployment of an immobile leaflet is possible, all measures should be ready for an immediate insertion of a valve in a valve. The fatal issue in our case was due to severe ventricular dilatation from AR